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ISSN:	0197-0186
Subjects:	Neurochemistry --
Description:	v. : ill. ; 25 cm.

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NEWS 4	JAN 27	A new search aid, the Company Name Thesaurus, available in CA/CAplus
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NEWS 17	May 19	PROUSDDR: One FREE connect hour, per account, in both May and June 2004
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=> s monocye () chemoattractant () protein-1
             0 MONOCYE
             1 MONOCYES
             1 MONOCYE
               (MONOCYE OR MONOCYES)
          8487 CHEMOATTRACTANT
          1702 CHEMOATTRACTANTS
          9385 CHEMOATTRACTANT
               (CHEMOATTRACTANT OR CHEMOATTRACTANTS)
       1632319 PROTEIN
       1126561 PROTEINS
       1892055 PROTEIN
               (PROTEIN OR PROTEINS)
       7921402 1
         29276 PROTEIN-1
               (PROTEIN(W)1)
L1           0 MONOCYE (W) CHEMOATTRACTANT (W) PROTEIN-1

=> s monocyte () chemoattractant () protein?
         33901 MONOCYTE
         26545 MONOCYTES
         42960 MONOCYTE
               (MONOCYTE OR MONOCYTES)
          8487 CHEMOATTRACTANT
          1702 CHEMOATTRACTANTS
          9385 CHEMOATTRACTANT
               (CHEMOATTRACTANT OR CHEMOATTRACTANTS)
       1930626 PROTEIN?
L2        4650 MONOCYTE (W) CHEMOATTRACTANT (W) PROTEIN?

=> s monocyte () chemoattractant () protein-1?
         33901 MONOCYTE
         26545 MONOCYTES
         42960 MONOCYTE
               (MONOCYTE OR MONOCYTES)
TERM '1?' EXCEEDED TRUNCATION LIMITS - SEARCH ENDED
```

```
You have entered a truncated stem which occurs in too many terms.
Make the stem longer and try again.  For example, if your original
term was 'degr?' to search for variations and the abbreviation for
'degradation', you could replace it with the expression '(degrdn OR
degrad?)'.  If your search term was numeric, e.g., 'C>5', reduce the
size of the range.

=> s monocyte () chemoattractant () protein-1
         33901 MONOCYTE
         26545 MONOCYTES
         42960 MONOCYTE
                 (MONOCYTE OR MONOCYTES)
          8487 CHEMOATTRACTANT
          1702 CHEMOATTRACTANTS
          9385 CHEMOATTRACTANT
                 (CHEMOATTRACTANT OR CHEMOATTRACTANTS)
       1632319 PROTEIN
       1126561 PROTEINS
       1892055 PROTEIN
                 (PROTEIN OR PROTEINS)
       7921402 1
         29276 PROTEIN-1
                 (PROTEIN(W)1)
L3        4357 MONOCYTE (W) CHEMOATTRACTANT (W) PROTEIN-1

=> s l3 and inhibitor?
        880882 INHIBITOR?
L4        1064 L3 AND INHIBITOR?

=> s l4 and glomeruler () nep?
             3 GLOMERULER
         79907 NEP?
             0 GLOMERULER (W) NEP?
L5           0 L4 AND GLOMERULER (W) NEP?

=> s l4 and lung () fibros?
        154872 LUNG
         39641 LUNGS
        168387 LUNG
                 (LUNG OR LUNGS)
         31792 FIBROS?
          1398 LUNG (W) FIBROS?
L6           4 L4 AND LUNG (W) FIBROS?

=> s l6 and review/dt
       1734809 REVIEW/DT
L7           0 L6 AND REVIEW/DT

=> s l6 and psoriasis
         10260 PSORIASIS
L8           1 L6 AND PSORIASIS

=> s l8 and review/dt
       1734809 REVIEW/DT
L9           0 L8 AND REVIEW/DT

=> s l8 and inflamm () bowel () disease
             4 INFLAMM
         11458 BOWEL
           186 BOWELS
```

```
         11596 BOWEL
                 (BOWEL OR BOWELS)
        686779 DISEASE
        191075 DISEASES
        776725 DISEASE
                 (DISEASE OR DISEASES)
             0 INFLAMM (W) BOWEL (W) DISEASE
L10          0 L8 AND INFLAMM (W) BOWEL (W) DISEASE

=> s l6 and inflammatory () bowel () disease
        121107 INFLAMMATORY
           255 INFLAMMATORIES
        121183 INFLAMMATORY
                 (INFLAMMATORY OR INFLAMMATORIES)
         11458 BOWEL
           186 BOWELS
         11596 BOWEL
                 (BOWEL OR BOWELS)
        686779 DISEASE
        191075 DISEASES
        776725 DISEASE
                 (DISEASE OR DISEASES)
          4183 INFLAMMATORY (W) BOWEL (W) DISEASE
L11          0 L6 AND INFLAMMATORY (W) BOWEL (W) DISEASE

=> s l6 and inflamm?
        182763 INFLAMM?
L12          4 L6 AND INFLAMM?

=> s l12 and review/dt
       1734809 REVIEW/DT
L13          0 L12 AND REVIEW/DT

=> d his

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     FILE 'HCAPLUS' ENTERED AT 11:38:50 ON 16 JUN 2004
L1           0 S MONOCYE () CHEMOATTRACTANT () PROTEIN-1
L2        4650 S MONOCYTE () CHEMOATTRACTANT () PROTEIN?
L3        4357 S MONOCYTE () CHEMOATTRACTANT () PROTEIN-1
L4        1064 S L3 AND INHIBITOR?
L5           0 S L4 AND GLOMERULER () NEP?
L6           4 S L4 AND LUNG () FIBROS?
L7           0 S L6 AND REVIEW/DT
L8           1 S L6 AND PSORIASIS
L9           0 S L8 AND REVIEW/DT
L10          0 S L8 AND INFLAMM () BOWEL () DISEASE
L11          0 S L6 AND INFLAMMATORY () BOWEL () DISEASE
L12          4 S L6 AND INFLAMM?
L13          0 S L12 AND REVIEW/DT

=> s l4 and inflamm?
        182763 INFLAMM?
L14        669 L4 AND INFLAMM?

=> s l14 and review/dt
       1734809 REVIEW/DT
L15         19 L14 AND REVIEW/DT
```

=> d l15, ibib abs, 1-19

L15 ANSWER 1 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:148100 HCAPLUS
DOCUMENT NUMBER: 140:368782
TITLE: Cardiovascular benefits of aldosterone receptor antagonists
AUTHOR(S): Williams, G. H.
CORPORATE SOURCE: Harvard Medical School, Brigham and Women's Hospital, Boston, MA, USA
SOURCE: Climacteric (2003), 6(Suppl. 3), 29-35
CODEN: CLIMFC; ISSN: 1369-7137
PUBLISHER: Taylor & Francis Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. There is considerable evidence in the setting of cardiovascular disease to suggest that, in addn. to the classic effects of aldosterone on sodium retention, blood vol., blood pressure and potassium homeostasis, aldosterone is involved in fibrotic end-organ damage by intermediate mechanisms involving an interplay between the mineralocorticoid receptor, sodium intake and a variety of mol. messengers. Such processes may help to explain the redn. in mortality that can be achieved in patients with severe heart failure and post-myocardial infarction by the addn. of an aldosterone receptor antagonist to std. therapy. Studies in animal models treated with the nitric oxide **inhibitor** No-nitro-L-arginine Me ester (L-NAME), angiotensin II and salt, with and without adrenalectomy, have demonstrated that myocardial damage can be eliminated by adrenalectomy or by administering an aldosterone receptor antagonist and is induced by adding back aldosterone to adrenalectomized animals. Importantly, at least a modest salt intake is an obligate co-factor. Other animal studies have established that an early stage in aldosterone-assocd. myocardial damage involves the release of proinflammatory mols., including cyclo-oxygenase type 2, osteopontin and **monocyte chemoattractant protein-1**. Taken together, these findings suggest that aldosterone in the presence of salt intake is a major cardiovascular risk factor mediated by **inflammatory** and fibrotic processes. Thus, mineralocorticoid receptor antagonists are likely to be effective addnl. agents to treat a broad range of cardiovascular diseases.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 2 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:119505 HCAPLUS
DOCUMENT NUMBER: 140:251067
TITLE: Modulators of urinary stone formation
AUTHOR(S): Khan, Saeed R.; Kok, Dirk J.
CORPORATE SOURCE: Department of Pathology, University of Florida, Gainesville, FL, USA
SOURCE: Frontiers in Bioscience (2004), 9, 1450-1482
CODEN: FRBIF6; ISSN: 1093-4715
URL: http://www.bioscience.org/2004/v9/af/1347/pdf.pdf
PUBLISHER: Frontiers in Bioscience
DOCUMENT TYPE: Journal; **General Review**; (online computer file)
LANGUAGE: English

AB A review. Urine contains compds. that modulate the nucleation, growth and aggregation of crystals as well as their attachment to renal epithelial

cells. These compds. may function to protect the kidneys against: 1, the possibility of crystn. in tubular fluid and urine, which are generally metastable with respect to calcium salts, 2, crystal retention within the kidneys thereby preventing stone formation and 3, possibly against plaque formation at the nephron basement membrane. Since oxalate is the most common stone type, the effect of various modulators on calcium oxalate (CaOx) crystn. has been examd. in greater details. Most of the **inhibitory** activity resides in macromols. such as glycoproteins and glycosaminoglycans while nucleation promotion activity is most likely sustained by membrane lipids. Nephrocalcin, Tamm-Horsfall protein, osteopontin, urinary prothrombin fragment 1, and bikunin are the most studied **inhibitory** proteins while chondroitin sulfate (CS), heparan sulfate (HS) and hyaluronic acid (HA) are the best studied glycosaminoglycans. Crystn. modulating macromols. discussed here are also prominent in cell injury, **inflammation** and recovery. Renal epithelial cells on exposure to oxalate and CaOx crystals produce some of the **inflammatory** mols. such as **monocyte chemoattractant protein-1** (MCP-1) with no apparent role in crystal formation. In addn., macrophages surround the CaOx crystals present in the renal interstitium. These observations indicate a close relationship between **inflammation** and nephrolithiasis.

REFERENCE COUNT: 324 THERE ARE 324 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 3 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:657221 HCAPLUS
DOCUMENT NUMBER: 139:212488
TITLE: **Inflammatory** cytokines and cardiovascular disease
AUTHOR(S): Ito, Takayuki; Ikeda, Uichi
CORPORATE SOURCE: Division of Cardiovascular Medicine, Jichi Medical School, Tochigi, 329-0498, Japan
SOURCE: Current Drug Targets: Inflammation & Allergy (2003), 2(3), 257-265
CODEN: CDTICU; ISSN: 1568-010X
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. The designation of atherosclerosis as a chronic **inflammatory** process represents an interesting paradigmatic shift for cardiologists. The plasma concns. of interleukin-6 and its hepatic byproduct, C-reactive protein, may reflect the intensity of occult plaque **inflammation** and the vulnerability to rupture. **Monocyte chemoattractant protein-1** and interleukin-8 play a crucial role in initiating atherosclerosis by recruiting monocytes/macrophages to the vessel wall, which promotes atherosclerotic lesions and plaque vulnerability. In addn., circulating levels of these proinflammatory cytokines increase in patients with acute myocardial infarction and unstable angina, but not in those with stable angina. Also, the plasma concns. of these cytokines increase after percutaneous coronary intervention, causing late restenosis after the procedure. Angiotensin II and other atherogenic factors induce these cytokines in the cardiovascular tissues through the activation of transcription factors, such as nuclear factor-κB or peroxisome proliferator-activated receptors. Conversely, HMG-CoA reductase **inhibitors** (statins) can potently inhibit these proinflammatory factors in the vessels. A small GTP-binding protein, Rho, may be a key mol. to explain the anti-**inflammatory** effects of statins. Interleukin-10 also exerts anti-**inflammatory** effects on the cardiovascular tissues, possibly

by deactivating proinflammatory cytokines and inducible nitric oxide synthase. Gene therapy using interleukin-10 may be a promising means for untreatable or complicated cases of cardiovascular diseases. Thus, therapeutic modulations of these **inflammatory** cytokines may be useful in the prevention of atherosclerosis and future cardiovascular events.

REFERENCE COUNT: 137 THERE ARE 137 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 4 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:521661 HCAPLUS
DOCUMENT NUMBER: 139:275277
TITLE: **Monocyte Chemoattractant Protein-1** (CCL2) in **Inflammatory** Disease and Adaptive Immunity: Therapeutic Opportunities and Controversies
AUTHOR(S): Daly, Christine; Rollins, Barrett J.
CORPORATE SOURCE: Department of Medical Oncology, Dana-Farber Cancer Institute, Boston, MA, 02115, USA
SOURCE: Microcirculation (New York, NY, United States) (2003), 10(3/4), 247-257
CODEN: MROCER; ISSN: 1073-9688
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review. Monocyte chemoattractant protein (MCP)-1 (CCL2) specifically attracts monocytes and memory T cells. Its expression occurs in a variety of diseases characterized by mononuclear cell infiltration, and there is substantial biol. and genetic evidence for its essential role in atherosclerosis and multiple sclerosis. Despite intensive screening, there are as yet no small-mol. antagonists of the receptor of MCP-1/CCL2, CCR2. However, biol. agents, including antibodies and **inhibitory** peptides, have been developed and may be useful for these indications. Recent evidence from genetically modified mice indicates that MCP-1 and CCR2 have unanticipated effects on T helper (Th) cell development. However, unlike the identical phenotypes of MCP-1/CCL2-/- and CCR2-/- mice in **inflammatory** diseases, the phenotypes of these mice are disparate in adaptive immunity: MCP-1 stimulates Th2 polarization, whereas CCR2 activation stimulates Th1 polarization. This presents both a challenge and an opportunity for targeting the MCP-1/CCL2/CCR2 axis in disease.
REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 5 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:493357 HCAPLUS
DOCUMENT NUMBER: 140:69932
TITLE: New insights into the treatment of pulmonary fibrosis
AUTHOR(S): Yurovsky, Vladimir V.
CORPORATE SOURCE: Department of Medicine, University of Maryland School of Medicine, Baltimore, MD, 21201, USA
SOURCE: Expert Opinion on Therapeutic Patents (2003), 13(7), 957-967
CODEN: EOTPEG; ISSN: 1354-3776
PUBLISHER: Ashley Publications Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review. Pulmonary fibrosis is a serious outcome of chronic lung

inflammation or environmental exposure. It is characterized by the replacement of lung epithelial tissues by fibroblasts in the repair process following lung injury and by excessive deposition of extracellular matrix that ultimately leads to a loss of functional gas exchange units. Current therapeutic strategies are aimed predominantly at suppressing lung **inflammation**, the role of which has been documented in the development of fibrosis. Data generated over recent years indicate that fibroproliferation and abnormalities in epithelial repair may have a greater pathophysiol. role than **inflammation**, thus representing new opportunities for therapeutic interventions. This review examines the patent literature in this area from 1999 to 2002 with some discussion of primary literature and older citations when appropriate.

REFERENCE COUNT: 106 THERE ARE 106 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 6 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:281476 HCAPLUS
DOCUMENT NUMBER: 139:127233
TITLE: Anti-**inflammatory** therapeutic strategy against atherosclerosis and restenosis after coronary intervention
AUTHOR(S): Kitamoto, Shiro; Egashira, Kensuke; Takeshita, Akira
CORPORATE SOURCE: Department of Cardiovascular Medicine, Graduate School of Medical Science, Kyushu University, Fukuoka, 812-8582, Japan
SOURCE: Journal of Pharmacological Sciences (Tokyo, Japan) (2003), 91(3), 192-196
CODEN: JPSTGJ; ISSN: 1347-8613
PUBLISHER: Japanese Pharmacological Society
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Atherosclerosis and restenosis after percutaneous coronary interventions have become major issues in public health in Western countries. Recent studies have revealed that **inflammation** plays an important role in pathogenesis of cardiovascular diseases. Vascular injury may involve an **inflammatory** response, which accelerates the recruitment and activation of monocytes through **monocyte chemoattractant protein-1** (MCP-1). MCP-1 expression has been shown to be increased in atherosclerotic lesions and balloon injured arteries. Recently, we have devised a new strategy for anti-MCP-1 gene therapy by transfecting mutant MCP-1 gene into skeletal muscle. This mutant MCP-1 has been shown to work as a dominant-neg. **inhibitor** of MCP-1. We here demonstrate that this strategy limited progression of pre-existing atherosclerotic lesions and improved the lesion compn. into a more stable phenotype in the hypercholesterolemic mice. This strategy also suppressed monocyte infiltration/activation in the injured site and markedly inhibited restenotic changes (neointimal hyperplasia) in the carotid artery in rabbits, rats, and monkeys after balloon injury or stent implantation. Therefore, MCP-1-mediated monocyte infiltration is essential in the development of restenotic changes as well as atherosclerosis progression. MCP-1 can be a practical therapeutic target for human restenosis and atherosclerosis.

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 7 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:201720 HCAPLUS
DOCUMENT NUMBER: 139:254552
TITLE: Chemistry and pharmacology of vascular protectants: A novel approach to the treatment of atherosclerosis and coronary artery disease
AUTHOR(S): Wasserman, Martin A.; Sundell, Cynthia L.; Kunsch, Charles; Edwards, David; Meng, Charles Q.; Medford, Russell M.
CORPORATE SOURCE: Department of Discovery Research, AtheroGenics, Inc., Alpharetta, GA, 30004, USA
SOURCE: American Journal of Cardiology (2003), 91(3A), 34A-40A
CODEN: AJCDAG; ISSN: 0002-9149
PUBLISHER: Excerpta Medica, Inc.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB This review addresses the role of oxidative stress in the pathol. of atherosclerosis and why it is now believed that atherosclerosis is not only a disease of oxidative stress but also of chronic **inflammation**. Perhaps more importantly, this review also describes the vascular protectant (V-protectant) technol. platform originated at AtheroGenics, Inc., from which a series of **inhibitory** compds. has emerged to treat a no. of chronic **inflammatory** diseases, including atherosclerosis. In atherosclerosis, these drugs not only act as antioxidants, but also as lipid modulators, **inhibitors** of **inflammation**, and **inhibitors** of gene expression. It is also important to understand the basis for considering vascular cell adhesion mol.-1 (VCAM-1) as a redn.-oxidn.-sensitive protein, which has a key role in the early phases of atherosclerosis. The review concludes with a description of the design and chem. of AtheroGenics' lead clin. development compd., AGI-1067, and an anal. of its preclin. in vitro and in vivo profile. AGI-1067 is a novel, potent antioxidant with anti-**inflammatory** properties. It inhibits gene expression of VCAM-1 and **monocyte chemoattractant protein-1**, decreases low-d. lipoprotein cholesterol levels, and prevents atherosclerosis in a no. of animal models. AGI-1067 is currently undergoing clin. trials as an antiatherosclerotic agent.
REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 8 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:151774 HCAPLUS
DOCUMENT NUMBER: 139:110891
TITLE: Molecular Mechanisms Mediating **Inflammation** in Vascular Disease
AUTHOR(S): Egashira, Kensuke
CORPORATE SOURCE: Graduate School of Medical Sciences, Department of Cardiovascular Medicine, Kyushu University, Fukuoka, Japan
SOURCE: Hypertension (2003), 41(3, Pt. 2), 834-841
CODEN: HPRTDN; ISSN: 0194-911X
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review. There are several clin. challenges for the treatment of intractable cardiovascular diseases, including restenosis, atherosclerotic complications resulting from plaque rupture, severe tissue ischemia, and heart failure. Emerging evidence suggests that an **inflammatory** process

is involved in the pathogenesis of such intractable diseases. In particular, **inflammatory** responses to arterial injury, which cause continuous recruitment and activation of monocytes mainly through activation of the **monocyte chemoattractant protein-1** (MCP-1) pathway, have a central role in restenosis and atherogenesis. We recently devised a new strategy for anti-MCP-1 therapy by transfecting an N-terminal deletion mutant of the MCP-1 gene into skeletal muscles. This mutant MCP-1 lacks the N-terminal amino acids 2 to 8, called 7ND, and works as a dominant-neg. **inhibitor** of MCP-1. We demonstrated that 7ND gene transfer suppresses monocyte infiltration/activation after arterial injury and markedly inhibits exptl. restenosis in animals after balloon injury or stent placement. Furthermore, 7ND gene transfer not only attenuated the development of early atherosclerotic lesions but also limited progression of preexisting atherosclerotic lesions and changed the lesion compn. into a more stable phenotype in hypercholesterolemic mice. Vascular **inflammation** mediated by MCP-1 might create a pos. feedback loop to enhance restenotic and atherosclerotic changes through activating lesional monocytes. Therefore, vascular **inflammation** mediated by MCP-1 has a central role in the development of exptl. restenosis, atherosclerosis, and plaque destabilization, leading to acute coronary syndrome. This strategy for gene therapy might be useful against human restenosis, thereby opening a new therapeutic window for antirestenosis and antiatherosclerosis paradigms.

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 9 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:629506 HCAPLUS
DOCUMENT NUMBER: 138:197976
TITLE: Pluripotential mechanisms of cardioprotection with HMG-CoA reductase **inhibitor** therapy
AUTHOR(S): Rosenson, Robert S.
CORPORATE SOURCE: Preventive Cardiology Center, Division of Cardiology, Departments of Medicine and Preventive Medicine, Northwestern University Medical School, Chicago, IL, USA
SOURCE: American Journal of Cardiovascular Drugs (2001), 1(6), 411-420
CODEN: AJCDDJ; ISSN: 1175-3277
PUBLISHER: Adis International Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Treatment with hydroxymethylglutaryl CoA (HMG-CoA) reductase **inhibitors** has been accompanied by a reduced risk of cardiovascular events. Rapid onset of clin. benefit and weak correlations between plasma low d. lipoprotein-cholesterol levels and coronary lumen change or cardiovascular events indicates that nonlipid mechanisms are involved in this beneficial effects with HMG-CoA reductase **inhibitors**. Furthermore, more rapid onset of clin. benefit with HMG-CoA reductase **inhibitors** in patients with acute coronary syndromes or acute myocardial infarction than in those with stable coronary heart disease suggest that HMG-CoA reductase **inhibitors** facilitate repair of ruptured or ulcerated atherosclerotic plaque, facilitate plaque stabilization and/or reduce thrombus formation on ruptured plaques. Treatment with HMG-CoA reductase **inhibitors** improved endothelial dysfunction in patients with hypercholesterolemia and this improvement in endothelial function was not correlated with redn. in total serum cholesterol levels. Similarly, redn. in endothelial pre-proendothelin mRNA expression and endothelin synthesis and blood

pressure lowering with HMG-CoA reductase **inhibitors** occurred independent of lipid-lowering. Finally, HMG-CoA reductase **inhibitors** increased endothelial nitric oxide levels i.e. upregulated endothelial nitric oxide synthetase expression via post-transcriptional mechanisms and prevented its down-regulation by oxidized LDL-C. HMG-CoA reductase **inhibitors** have been shown to modulate the immune response by inhibiting activation of immune-competent cells such as macrophages, and antigen presentation to macrophages by T cells. Treatment with HMG-CoA reductase **inhibitors** can reduce expression, prodn. and circulating levels of chemokines (**monocyte chemoattractant protein-1**) and proinflammatory cytokines [tumor necrosis factorα, interleukin (IL)-6 and IL-1β]. HMG-CoA reductase **inhibitors** reduced **inflammation** in human atheroma: significantly fewer macrophages and T cells, less oxidized LDL-C and higher collagen content. In addn., treatment with HMG-CoA reductase **inhibitor** led to decreased cell death within the atheroma. Treatment with these agents also reduced expression of inducible cellular adhesion mols., decreased secretion of metalloproteinases by macrophages, reduced vascular smooth muscle cell apoptosis. Lastly, HMG-CoA reductase **inhibitors** appear to have important effects on the thrombogenesis: reduced expression of tissue factor prodn. and activity; increased prodn. of tissue factor package **inhibitor**; decreased platelet thrombus formation and improved fibrinolysis as a result of lowered plasminogen activator **inhibitor**-1 levels. As the pluripotential cardioprotective mechanisms of HMG-CoA reductase **inhibitors** are further elucidated, it is envisaged that treatment with HMG-CoA reductase **inhibitors** will be initiated earlier and more frequently in patients with hypercholesterolemia.

REFERENCE COUNT: 90 THERE ARE 90 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 10 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:579996 HCAPLUS
DOCUMENT NUMBER: 137:320372
TITLE: Angiotensin II as a pro-**inflammatory** mediator
AUTHOR(S): Phillips, M. Ian; Kagiyama, Shuntaro
CORPORATE SOURCE: Departments of Physiology and Functional Genomics College of Medicine, University of Florida, Gainesville, FL, 32610, USA
SOURCE: Current Opinion in Investigational Drugs (PharmaPress Ltd.) (2002), 3(4), 569-577
CODEN: COIDAZ; ISSN: 1472-4472
PUBLISHER: PharmaPress Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Angiotensin II (Ang II), the most important component of the renin-angiotensin system, is usually assocd. with hypertension and renal failure. Through its pro-**inflammatory** actions, it also plays an important role in each step of the development of atherosclerotic plaques and plaque rupture. Ang II stimulates the expression of nuclear factor-κB (NFκB), a transcription factor which regulates gene expression of **inflammatory** cytokines such as interleukin-6 (IL-6) and **monocyte chemoattractant protein-1** (MCP-1). Ang II type 1 receptors (AT1) and angiotensin converting enzyme (ACE) are dramatically increased in atherosclerotic plaques, particularly in monocytes at the fibrous cap. Thus, in multiple ways, Ang II is a crit. factor in atherosclerotic plaque formation, **inflammation** and plaque stability. ACE **inhibitors** and AT1R **inhibitors** could therefore be appropriate therapeutic agents in the treatment of atherosclerosis.

REFERENCE COUNT: 90 THERE ARE 90 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 11 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:490209 HCAPLUS
DOCUMENT NUMBER: 137:260778
TITLE: Clinical importance of endothelial function in arteriosclerosis and ischemic heart disease
AUTHOR(S): Egashira, Kensuke
CORPORATE SOURCE: Department of Cardiovascular Medicine, School of Medical Sciences, Kyushu University, Fukuoka, Japan
SOURCE: Circulation Journal (2002), 66(6), 529-533
CODEN: CJIOBY; ISSN: 1346-9843
PUBLISHER: Japanese Circulation Society
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. The vascular endothelium is a dynamic endocrine organ that regulates vascular tone, local homeostasis, and the fibro-**inflammatory**-proliferative process. These responses are mediated by various substances released from the endothelium in response to physiol. stimuli, including prostacyclin, endothelin and, most importantly, nitric oxide (NO). NO mediates vasodilation and inhibits platelet aggregation, thrombus formation, expression of adhesion mols. and chemokines for leukocytes, and oxidative stress. It also attenuates growth and proliferation of vascular smooth muscle cells. Risk factors for atherosclerosis, such as hypercholesterolemia, hypertension, diabetes and cigarette smoking, impair endothelial function, which leads to atherosclerosis and results in ischemic manifestations such as acute coronary syndrome and stroke. Thus, therapeutic intervention aimed at increasing NO bioavailability by statins or angiotensin-converting enzyme **inhibitors** might improve patient prognosis. Vascular endothelial function is an important and clin. relevant therapeutic target for cardiovascular disease.

REFERENCE COUNT: 80 THERE ARE 80 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 12 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:155814 HCAPLUS
DOCUMENT NUMBER: 137:246072
TITLE: Cytokine in heart failure and phosphodiesterase **inhibitors**
AUTHOR(S): Yomogida, Shinichi; Endo, Masao
CORPORATE SOURCE: School of Medicine, Yamagata University, Japan
SOURCE: Junkan Seigyo (2001), 22(4), 351-355
CODEN: JUSEE7; ISSN: 0389-1844
PUBLISHER: Nippon Junkan Seigyo Igakkai
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: Japanese

AB A review. Expression of proinflammatory cytokines and chemokines in heart failure and effects of phosphodiesterase **inhibitors** on proinflammatory cytokine expression are discussed. The topics discussed are (1) expression of tumor necrosis factor-α (TNF-α), macrophage **inflammatory** protein-1α (MIP-α), and **monocyte chemoattractant protein-1** (MCP-1) in heart failure and (2) phosphodiesterase isoenzymes and effects of phosphodiesterase **inhibitors** Amrinone and Pimobendan on TNF-α, IL-1β, and nitric oxide prodn.

L15 ANSWER 13 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:891885 HCAPLUS
DOCUMENT NUMBER: 136:15338
TITLE: Cytokines and chemokines: mediators for intercellular communication in the brain
AUTHOR(S): Minami, Masabumi
CORPORATE SOURCE: Department of Molecular Pharmacology, Faculty of Pharmaceutical Sciences, Kyoto University, 46-29 Yoshida Shimoadachi-cho, Sakyo-ku, Kyoto, 606-8501, Japan
SOURCE: Yakugaku Zasshi (2001), 121(12), 875-885
CODEN: YKKZAJ; ISSN: 0031-6903
PUBLISHER: Pharmaceutical Society of Japan
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: Japanese

AB A review. The brain includes glial cells (astrocytes, microglia and oligodendrocytes) and endothelial cells in addn. to neurons. Under some pathol. conditions, it is invaded by leukocytes such as neutrophils, monocytes/macrophages and lymphocytes. Intercellular communication across these cell species is supposed to play crucial roles both in the brain functions and dysfunctions. However, the mol. basis of such intercellular communication remains unclear. We have studied the roles of cytokines and chemokines, which have been investigated as essential mediators in the immune and **inflammatory** systems, in intercellular communication across neurons. glial cells, endothelial cells and leukocytes. MRNA expression of cytokines such as interleukin-1β was induced in brain microglia by i.p. injection of excitotoxin and neurostimulant, at least, partly via catecholaminergic systems. MRNA of other cytokines such as leukemia **inhibitory** factor was induced in astrocytes. This cytokine specifically induced nociceptin mRNA in the cultured cortical neurons. Constitutive expression of some chemokines such as fractalkine and stromal cell derived factor-1α was obsd. in the brain, suggesting that they play important roles in maintenance of brain homeostasis or detn. of the patterning of neurons and/or glial cells in the developing and adult brains. Cytokines such as interleukin-1, and chemokines such as **monocyte chemoattractant protein-1** and macrophage **inflammatory** protein-1α were produced in ischemic brain and implicated in ischemic brain injury. In addn. to ischemia, cytokines, chemokines and their receptors have been shown to be involved in various neurodegenerative diseases such as multiple sclerosis, Alzheimer's disease and AIDS dementia syndrome. They are potential targets for therapeutic intervention for neurodegenerative diseases.

L15 ANSWER 14 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:506438 HCAPLUS
DOCUMENT NUMBER: 135:282560
TITLE: **Inhibitors** of **monocyte chemoattractant protein-1**/CC ligand 2 and its receptor CCR2
AUTHOR(S): Howard, O. M. Zack; Yoshimura, Teizo
CORPORATE SOURCE: Laboratory of Molecular Immunoregulation, Center for Cancer Research, National Cancer Institute-Frederick, Frederick, MD, 21702-1201, USA
SOURCE: Expert Opinion on Therapeutic Patents (2001), 11(7), 1147-1151
CODEN: EOTPEG; ISSN: 1354-3776

PUBLISHER: Ashley Publications Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review with refs. Chemoattractant cytokines (chemokines) have been shown to be pro-**inflammatory** and are thus likely targets for therapeutic intervention. An agent that interferes with directed migration of leukocytes to an **inflammatory** site is potentially a candidate anti-**inflammatory** drug. A specific chemokine, monocyte chemoattractant protein (MCP)-1 or CC ligand 2 (CCL2), and its receptor, CC-chemokine receptor 2 (CCR2), have been implicated in both acute and chronic **inflammatory** and autoimmune diseases assocd. with infiltration of monocytes, macrophages, dendritic cells, NK cells, basophils and memory T-cells. Genetic modification of CCL2 and CCR2 in murine models has demonstrated the potential for antagonists to prevent atherogenic vascular disease and autoimmune **inflammatory** diseases. Modified CCL2 peptides, which still bind but no longer activate CCR2, demonstrated the therapeutic potential of CCL2 **inhibitors** in animal models of arthritis. Several classes of small mol. wt. CCL2 **inhibitors** have also been shown to inhibit chemotaxis in response to CCL2 in vitro and in animal models. However, more work is needed to establish the clin. efficacy of these CCL2 **inhibitors**.
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 15 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:51694 HCAPLUS
DOCUMENT NUMBER: 135:3621
TITLE: How renal cytokines and growth factors contribute to renal disease progression
AUTHOR(S): Benigni, Ariela; Remuzzi, Giuseppe
CORPORATE SOURCE: Mario Negri Institute for Pharmacological Research, Bergamo, 24125, Italy
SOURCE: American Journal of Kidney Diseases (2001), 37(1, Suppl. 2), S21-S24
CODEN: AJKDDP; ISSN: 0272-6386
PUBLISHER: W. B. Saunders Co.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review with 20 refs. Terminal renal failure is the final common fate of chronic nephropathies regardless of the type of original insult. After removal of a crit. no. of nephrons, adaptive hemodynamic changes in the remaining nephrons ensure enough filtration power to the kidney but are ultimately detrimental. Such changes are largely mediated by the local formation of angiotensin II (AII) and prevented by the use of angiotensin-converting enzyme **inhibitors**, which also limit the forced opening of large unselective pores in the glomerular barrier, restoring size selectivity. Recent studies suggested that proteins filtered through the glomerular capillary, previously considered a marker of the severity of renal lesions, might have intrinsic toxicity on the proximal tubular cells and a contributory role in the progression of renal damage. Protein overload of proximal tubular cells induced the secretion of endothelin-1 (ET-1), **monocyte chemoattractant protein-1** (MCP-1), and regulated on activation, normal T expressed and secreted (RANTES) that was mainly directed toward the basolateral compartment of the cell. Evidence available in rat models of proteinuric renal disease shows that expression of genes encoding such vasoactive and proinflammatory mols. as ET-1, MCP-1, and RANTES was consistently upregulated, and synthesis of the corresponding peptides was enhanced in renal tissue. Addnl. mechanisms of

proximal tubular cell activation leading to interstitial **inflammation** and matrix deposition are the filtration of protein-bound metals and hormones and deposition and activation of filtered complement. Limiting protein traffic and the biol. effect of excessive tubular protein reabsorption by drugs interfering with AII synthesis or biol. activity prevents renal disease progression.

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 16 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1999:674509 HCAPLUS
DOCUMENT NUMBER: 131:309326
TITLE: Oxidized low density lipoprotein. Atherogenic and proinflammatory characteristics during macrophage foam cell formation. An **inhibitory** role for nutritional antioxidants and serum paraoxonase
AUTHOR(S): Kaplan, Marielle; Aviram, Michael
CORPORATE SOURCE: Lipid Research Laboratory, Bruce Rappaport Faculty Medicine, Rappaport Family Institute Research Medical Sciences, Technion, Haifa, 31096, Israel
SOURCE: Clinical Chemistry and Laboratory Medicine (1999), 37(8), 777-787
CODEN: CCLMFW; ISSN: 1434-6621
PUBLISHER: Walter de Gruyter GmbH & Co. KG
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with 122 refs. is given. Oxidative stress and **inflammatory** processes are of major importance in atherogenesis because they stimulate oxidized LDL (Ox-LDL)-induced macrophage cholesterol accumulation and foam cell formation, the hallmark of early atherosclerosis. Under oxidative stress, both blood monocytes and plasma lipoproteins invade the arterial wall, where they are exposed to atherogenic modifications. Oxidative stress stimulates endothelial secretion of **monocyte chemoattractant protein 1** (MCP-1) and of macrophage colony stimulating factor (M-CSF), leading to monocyte adhesion and differentiation, resp. LDL binds to extracellular matrix (ECM secreted by endothelial cells, smooth muscle cells and macrophages) proteoglycans, in a process that contributes to the enhanced susceptibility of the lipoprotein to oxidn. by arterial wall macrophages. ECM-retained Ox-LDL is taken up by activated macrophages via their scavenger receptors. This leads to cellular cholesterol accumulation and enhanced atherogenesis. Protection of LDL against oxidn. by antioxidants that can act directly on the LDL, or indirectly on the cellular oxidative machinery, or conversion of Ox-LDL to a non-atherogenic particle by HDL-assocd. paraoxonase (PON-1), can contribute to attenuation of atherosclerosis.

REFERENCE COUNT: 122 THERE ARE 122 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 17 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1999:55502 HCAPLUS
DOCUMENT NUMBER: 130:250265
TITLE: Angiotensin II is involved in the progression of renal disease: importance of non-hemodynamic mechanisms
AUTHOR(S): Wolf, G.
CORPORATE SOURCE: Department of medicine, division of nephrology and

osteology, University of Hamburg, Germany
SOURCE: Nephrologie (1998), 19(7), 451-456
CODEN: NEPHDY; ISSN: 0250-4960
PUBLISHER: Medecine et Hygiene
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review, with 51 refs. Several recent studies have provided clear evidence that angiotensin-converting enzyme (ACE)-**inhibitors** slow the progression of renal disease. These effects are mainly independent from a comitant redn. in systemic blood pressure. Thus, angiotensin II (Ang II) exerts other effects on the kidney which are involved in the loss of renal function. Ang II induces proliferation of cultured mesangial and glomerular endothelial cells. Our group was the first to demonstrate that Ang II stimulates hypertrophy of cultured proximal tubular cells. Ang II stimulates bioactivation and expression of transforming growth factor-β (TGF-β) in tubular MCT cells. This Ang II-mediated expression of TGF-β is due to an increase in transcriptional activity. A neutralizing anti-TGF-β antibody attenuates the Ang II-induced increase in protein synthesis in MCT cells suggesting that the hypertrophy is mediated by synthesis and activation of endogenous TGF-β. Proximal tubular cells undergoing Ang II-mediated hypertrophy are arrested in the G1-phase of the cell cycle and express typical G1-phase-assocd. genes. Induction of such G1-phase-assocd. early growth response genes have been also described in vivo after infusion of Ang II into the renal artery. This G1-phase arrest depends on the induction of the cyclin-dependent kinase (CdK) **inhibitor** p27Kip1. P27Kip1 expression is stimulated after incubation of LLC-PK1 cells with Ang II or TGF-β and binds to cyclin D1-CdK4 complexes, inhibits their kinase activity, and hampers G1-phase exit. Ang II stimulates transcription of collagen type IV in MCT cells. In addn. to the classical a1 (IV) chain, a3 (IV) collagen, which has normally a restricted localization in the kidney, is also induced. This stimulation is mediated by endogenous synthesis and autocrine action of TGF-β because a neutralizing anti-TGF-β antibody as well as TGF-β antisense oligonucleotides attenuate Ang II-induced collagen type IV transcription and synthesis. In addn., Ang II exerts immunomodulatory effects on the kidney through the induction of chemokines such as MCP-1 and RANTES. In conclusion, Ang II has emerged as a multifunctional acting as a growth factor and a profibrogenic cytokine, and even having **inflammatory** properties.

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 18 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1998:440575 HCAPLUS
DOCUMENT NUMBER: 129:215276
TITLE: Will MCP-1 and RANTES take center stage in **inflammatory** diseases including asthma?
AUTHOR(S): Conti, Pio; Barbacane, Renato C.; Di Gioacchino, Mario; Reale, Marcella
CORPORATE SOURCE: Division of Immunology, Department of Oncology and Neurosciences, University of Chieti School of Medicine, Chieti, 66100, Italy
SOURCE: Allergy and Asthma Proceedings (1998), 19(3), 121-123
CODEN: AAPRFV; ISSN: 1088-5412
PUBLISHER: OceanSide Publications, Inc.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with 27 refs. RANTES and MCP-1 are potent pro-**inflammatory** cytokines that can chemoattract mast cells in addn. to other **inflammatory** cells. Recent studies show that RANTES and MCP-1 may increase the no. of mast cell migration in bronchial mucosa during asthma. Therefore, an **inhibitory** effect of RANTES and MCP-1 could play a role in controlling the **inflammatory** response in asthma and other **inflammatory** diseases.

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 19 OF 19 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1996:134893 HCAPLUS
DOCUMENT NUMBER: 124:199776
TITLE: Cytokines regulate vascular functions related to stability of the atherosclerotic plaque
AUTHOR(S): Libby, Peter; Sukhova, Galina; Lee, Richard T.; Galis, Zorina S.
CORPORATE SOURCE: Department Medicine, Brigham & Women's Hospital and Harvard Medical School, Boston, MA, 02115, USA
SOURCE: Journal of Cardiovascular Pharmacology (1995), 25(Suppl. 2), S9-S12
CODEN: JCPCDT; ISSN: 0160-2446
PUBLISHER: Lippincott-Raven
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review, with 23 refs. The cytokines are multipotent mediators of **inflammation** and immunity that can affect key functions of vascular wall cells. Growing evidence suggests that cytokines participate as autocrine or paracrine mediators in atherogenesis, as cells in lesions can both produce and respond to these mediators. The functions of vascular wall cells regulated by cytokines may influence lesion initiation, progression, or complication. For example, cytokines can regulate the expression of adhesion mols. crucial to the recruitment of leukocytes to lesions, including vascular cell adhesion mol.-1 (VCAM-1). Cytokines such as interleukin-1 (IL-1) and tumor necrosis factor-α (TNF-α) can regulate the prodn. of **monocyte chemoattractant protein-1** (MCP-1), a potential signal for directed migration of monocytes into the intima. Cytokines can also regulate genes that encode other growth factors and cytokines themselves. TNF-α can induce IL-1 mRNA in human endothelial (EC) and smooth-muscle cells (SMC). IL-1 and TNF-α can augment the prodn. by vascular cells of macrophage-colony stimulating factor (M-CSF), which may promote growth and activation of mononuclear phagocytes. Cytokines can exert both pro- and antiatherogenic actions. Activated T cells in human atheroma may secrete the lymphokine IFN-γ, an **inhibitor** of SMC proliferation. Cytokines influence vasomotor tone in arteries, e.g., by inducing a form of nitric oxide synthase, the enzyme that synthesizes the vasodilatory nitric oxide radical. The cytokines also modulate endothelial functions that govern the formation and stability of blood thrombi. Finally, in the late stags of the disease, matrix metalloproteinases derived from macrophages or smooth-muscle cells themselves may contribute to weakening of the fibrous cap in the vulnerable shoulder area, promoting plaque rupture and occlusive thrombosis, culminating in the dramatic clin. manifestations of atherosclerosis, including myocardial infarction and stroke. Thus, cytokines can influence multiple aspects of atherogenesis and provide new and interesting targets for therapeutic intervention.

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                     CA/CAplus
NEWS  5   FEB 05     German (DE) application and patent publication number format
                     changes
NEWS  6   MAR 03     MEDLINE and LMEDLINE reloaded
NEWS  7   MAR 03     MEDLINE file segment of TOXCENTER reloaded
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NEWS 15   APR 27     NLDB: New search and display fields available
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NEWS 17   May 19     PROUSDDR: One FREE connect hour, per account, in both May
                     and June 2004
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NEWS 19   May 12     Polymer links for the POLYLINK command completed in REGISTRY
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                     Conference
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                     SDIs in CAplus
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              MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
              AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004
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FILE COVERS 1907 - 16 Jun 2004 VOL 140 ISS 25
FILE LAST UPDATED: 15 Jun 2004 (20040615/ED)

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substance identification.

```
=> s rantes () inhibitor?
          3405 RANTES
        880882 INHIBITOR?
L1           3 RANTES (W) INHIBITOR?

=> s l1 and review/dt
       1734809 REVIEW/DT
L2           1 L1 AND REVIEW/DT

=> d l2, ibib abs, 1

L2   ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2004 ACS on STN
```

Full Text | Citing References

ACCESSION NUMBER: 1997:712630 HCAPLUS
DOCUMENT NUMBER: 127:357799
TITLE: High throughput screening for identification of RANTES chemokine expression inhibitors
AUTHOR(S): Barnes, Debra A.; Jones, Steven W.; Perez, H. Daniel
CORPORATE SOURCE: USA
SOURCE: Methods in Enzymology (1997), 287(Chemokines), 292-304
CODEN: MENZAU; ISSN: 0076-6879
PUBLISHER: Academic
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review with 31 refs. on a procedure using CH235 astrocytoma cells to screen for inhibitors of RANTES expression.

```
=> s MCP-1 () inhibitor?
          7067 MCP
           380 MCPS
          7227 MCP
                 (MCP OR MCPS)
       7921402 1
          3461 MCP-1
```

```
                (MCP(W)1)
        880882 INHIBITOR?
L3          14 MCP-1 (W) INHIBITOR?

=> s l3 and review/dt
      1734809 REVIEW/DT
L4           1 L3 AND REVIEW/DT

=> d l4, ibib abs, 1

L4   ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2004 ACS on STN
```

Full Text | Citing References

```
ACCESSION NUMBER:          2003:704642  HCAPLUS
DOCUMENT NUMBER:           139:285453
TITLE:                     AGI-1067: Treatment of atherosclerosis VCAM-1 and
                           MCP-1 expression inhibitor antioxidant
AUTHOR(S):                 Sorbera, L. A.; Castaner, J.
CORPORATE SOURCE:          Prous Science, Barcelona, 08080, Spain
SOURCE:                    Drugs of the Future (2003), 28(5), 421-424
                           CODEN: DRFUD4; ISSN: 0377-8282
PUBLISHER:                 Prous Science
DOCUMENT TYPE:             Journal; General Review
LANGUAGE:                  English
AB   A review.  AGI-1067 is a monosuccinate ester of probucol that exhibited
     marked lipid-lowering and antioxidant activity.  AGI-1067 potently
     inhibited VCAM-1 and MCP-1 expression and smooth muscle cell proliferation
     and was effective in animal models of atherosclerosis and hyperlipidemia.
     The agent has shown efficacy in the prevention of atherosclerosis in
     patients with coronary artery disease and in preventing restenosis in
     patients undergoing percutaneous coronary interventions.  AG-1067 is
     currently undergoing phase III trials with an indication for secondary
     prevention of atherosclerotic cardiovascular disease.
REFERENCE COUNT:           22    THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS
                                 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> S Rantes () chemotaxis?
          3405 RANTES
         13976 CHEMOTAXIS?
L5           8 RANTES (W) CHEMOTAXIS?

=> s l5 and inhibitors
        463870 INHIBITORS
L6           1 L5 AND INHIBITORS

=> s l6 and review/dt
       1734809 REVIEW/DT
L7           0 L6 AND REVIEW/DT

=> s  MCP-1 () chemotaxis?
          7067 MCP
           380 MCPS
          7227 MCP
                 (MCP OR MCPS)
       7921402 1
          3461 MCP-1
                 (MCP(W)1)
         13976 CHEMOTAXIS?
L8           1 MCP-1 (W) CHEMOTAXIS?
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=> s 18 and review/dt
       1734809 REVIEW/DT
L9           0 L8 AND REVIEW/DT

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